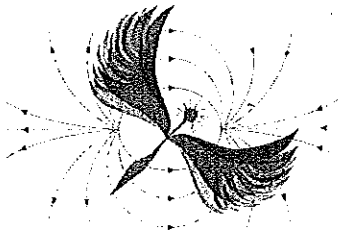

March 2, 2009

Dear Respected Legislators,

I am writing today in support of **HB 6572: An Act Banning Bisphenol-A in Children's Products and Food Products and Prohibiting Certain Alterative Substances.** I earned my PhD in the laboratory of Drs. Ana Soto and Carlos Sonnenschein at Tufts University School of Medicine; my doctoral dissertation examined the effects of perinatal BPA exposure on the development of the mammary gland. I am an author on five primary research articles and three reviews on Bisphenol-A, all of which have been peer reviewed. I served as the lead author on the most comprehensive review of the Bisphenol A human exposure to date. I also signed the Chapel Hill Consensus Statement, a product of the National Institutes of Environmental Health Science's expert panel on Bisphenol-A. Because of my expertise in the study of human exposures to Bisphenol-A, I have been invited to serve on a panel of experts to generate written regulatory guidance at the German Federal Environment Agency, the Umweltbundesamt, equivalent to the US EPA.

I am providing you with scientific information about bisphenol A. I have tried to keep these comments brief while still providing you with enough information to understand the significance of your current undertaking. Please feel free to contact me if I can be of further help to your committee.

Sincerely,

Laura N. Vandenberg

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Written Testimony of Laura N. Vandenberg, PhD, Tufts University

Before the Connecticut General Assembly Environment Committee, March 2nd, 2009

Testimony in Support of:

Raised HB No. 6572: An Act Banning Bisphenol-A in Children's Products and Food Products and Prohibiting Certain Alterative Substances.

General Information about BPA

Bisphenol A (BPA) is one of the highest volume chemicals produced worldwide with over 6 billion pounds produced each year and over 100 tons released into the atmosphere by yearly production (Markey 2001a). It is the building block of polycarbonate plastic. Numerous studies have found that BPA leaches from polycarbonate baby bottles (Sun 2000, Brede 2003, Wong 2005) and re-usable water bottles (Le 2008). Other polycarbonate containers (e.g., Tupperware) intended to be used as reusable food containers, food-contact items such as polyvinyl chloride stretch films, and some papers and cardboards used as food containers have been examined for their BPA content (reviewed in Vandenberg 2007a). BPA is also used as a component in epoxy resins that are used to coat the insides of food cans; several studies have documented conditions that support or enhance BPA migration from these coatings (Brotons 1994, Takao 2002, Kang 2003). Many studies have also examined BPA levels leaching from epoxy resins to foods stored in cans (Yoshida 2001, Goodson 2002). Still others have found BPA contamination in canned infant formula (Biles 1997, Kuo 2004).

Very few studies have estimated total BPA exposure from multiple sources. Using data from contamination in the environment (water, air, soil) and food contamination (can surfaces, plastic containers), the daily human intake of BPA was estimated at less than 1 µg/kg BW/day (Kang 2006). Alternatively, the European Commission's Scientific Committee on Food estimated BPA exposure to be 0.48-1.6 µg/kg BW/day from food sources, while Thompson et al. estimated that New Zealanders consume as much as 4.8 µg/day from dietary sources alone (EU 2003, Thompson 2003).

Two studies were conducted to estimate BPA exposure levels in young children. The first was designed to examine their potential exposures at home and in daycare (Wilson 2003). BPA was detected in indoor and outdoor air samples, floor dust and play area soil, and in liquid and solid foods in both locations at similar levels. A second observational study examined BPA exposures in 257 preschool children (Wilson 2007). This study verified that BPA could be found in more than 50% of indoor air, hand wipe, solid food and liquid food samples and suggested that 99% of exposures of preschool children originated in the diet. Dozens of additional studies have demonstrated that BPA can be found in dust samples, indoor and outdoor air, sewage leachates, and water samples (including drinking water) from around the world (reviewed in Vandenberg 2007a).

1. BPA has been measured in many human samples

Since 1999 (Sajiki 1999), more than a dozen studies using a variety of different analytical techniques have measured free, unconjugated BPA concentrations in human blood at levels ranging from 0.2–20 ng/ml serum (see Vandenberg 2007a). Several

studies report the relatively high levels of BPA in the blood of pregnant women, umbilical cord blood, and fetal plasma (Ikezuki 2002, Schonfelder 2002, Yamada 2002, Tan 2003), which indicates that BPA crosses the maternal-fetal placental barrier. BPA has also been measured in human urine from several populations around the world. These studies confirm widespread human exposure to BPA, as suspected from the studies of BPA in blood. A 2005 study conducted by the US Centers for Disease Control and Prevention (CDC) detected BPA in 95% of urine samples from a reference population of 394 American adults using isotope dilution GC-MS with average levels of total BPA in male and female urine of 1.63 and 1.12 ng/ml, respectively (Calafat 2005). A more recent CDC study of over 2500 Americans supports this finding, with BPA detected in 92.6% of participants (Calafat 2008a). Measured urine concentrations ranged from 0.4-149 µg/L with a geometric mean of 2.6 µg/L and were significantly higher in children and adolescents compared to adults.

A recent study (Edginton 2008) used pharmacokinetic modeling tools to predict blood concentrations of BPA in neonates, young infants, children and adults. These models suggest that if newborns are exposed to the same dose of BPA as adults (adjusted for differences in body weight), the amount of BPA that will remain circulating in the blood will be 10 times higher in the newborn. Neonates and infants simply lack the metabolic machinery to remove BPA from their blood circulation. These models were subsequently supported by another recent study conducted by the CDC examining neonates and infants in the Neonatal Intensive Care Unit (NICU) (Calafat 2008b). The authors found that BPA concentrations were almost 10 times higher in urine collected from NICU babies than in the general population.

2. A few limited epidemiology studies have connected BPA exposure with human diseases

Human studies of possible health effects of BPA exposure are extremely limited. BPA levels in blood have been associated with a variety of conditions in women including obesity, problems with the uterine lining, recurrent miscarriages, and polycystic ovarian syndrome (PCOS). Two studies found that women with PCOS had higher blood levels of BPA than women without PCOS (Takeuchi 2002, Takeuchi 2004). Three studies found higher BPA exposure for health-related outcomes that are associated with chromosomal abnormalities. One study found higher maternal serum BPA among women carrying fetuses with abnormal chromosome numbers compared to women carrying fetuses with normal chromosomes (Yamada 2002). In another epidemiology study, an association between serum BPA levels and recurrent miscarriage was reported (Sugiura-Ogasawara 2005); average BPA levels were more than three times as high in women with a history of three or more consecutive first-trimester miscarriages compared to women without fertility problems that had never been pregnant. Additionally, among women that later became pregnant, there was some evidence of lower BPA levels among the women who subsequently had a successful pregnancy as compared to those that miscarried again.

At this time, only one large and well-controlled study of the possible health effects of BPA exposure on humans has been conducted, revealing positive correlations between urinary BPA concentrations and the prevalence of diabetes, heart disease and liver toxicity (Lang 2008). This cross-sectional study was performed using samples and information collected for the CDC study and included 1455 American adults. However, additional studies are needed to determine whether the associations between BPA concentrations in urine and disease prevalence are causal.

There are a few problems with determining the true impact of BPA exposure on human health. First, to do a good study, scientists need to control the amount of BPA

each subject is exposed to, which is impossible considering that over 90% of Americans are exposed to significant levels in their normal lives. Thus, it is nearly impossible to find a good "control" group that is completely unexposed to compare to any research subjects. Second, many epidemiology studies are conducted retrospectively, i.e. by asking research subjects about what happened in their past, and comparing that to their present health conditions. Unfortunately, virtually all people on earth were exposed to plastics in their past, and very few will have knowledge more specific than that. Finally, because scientists are most concerned about BPA exposure during early development, a good human study to address the effects of fetal/neonatal exposure to BPA would require dosing pregnant and nursing women to BPA. Because of the known effects of BPA on animals, this study would be unethical, and it would be very difficult to recruit informed research subjects.

For this reason, BPA researchers have used animal models to study the effects of BPA exposure during early development (see Richter 2007 for a complete review).

3. Animal studies illustrate significant connections between BPA exposure and disease

Animal studies have been used to study the effects of exposure to BPA during specific periods of development. Over 140 studies have examined animals exposed to BPA at concentrations at or below the level that is considered "safe" by the EPA.

ii. Behavior & brain

BPA-exposed animals demonstrated a loss of sex differences in the number of dopamine neurons in the dimorphic anteroventral periventricular nucleus of the hypothalamus; this was due to a defeminization of the exposed females (Rubin 2006). What this means is that a specific brain region that is normally different in males and females looks similar in animals exposed to BPA during fetal and neonatal development. The female brain looked like the male brain. This is expected to have drastic impacts on the ability of females to have normal estrus cycles (the rodent equivalent of menstrual cycles) and may prevent these females from getting pregnant.

Several studies have examined the impact of low dose BPA exposure during early development on behavior at puberty and in adulthood. Because hormones play a role in establishing different patterns of behavior in males and females, behavioral experiments on BPA have focused on social and sexual behaviors that are known to be different in male and female rodents. Studies from several groups have illustrated that BPA exposure obliterates sex differences in behavior (Dessi-Fulgheri 2002, Rubin 2006, Fujimoto 2006).

iii. Male reproduction

Low dose BPA exposure during perinatal development led to alterations of the organs of the male reproductive tract including changes in testis weight at puberty and in adulthood (Kabuto 2004, Kawai 2003). Perhaps most interesting are the alterations induced by BPA in the prostate. Fetal BPA exposure induced increased prostate size in adults (Welshons 1999) and also altered specific aspects of the prostate tissue that may affect its function (Ramos 2001). Further studies of fetal BPA exposure revealed that increases in prostate size could be detected in the fetus (Timms 2005).

iv. Female reproduction

Female reproductive endpoints, including the rate of reproductive functions, are affected by BPA exposure as well. BPA exposure affects the timing of puberty (Howdeshell 1999, Honma 2002). Alterations were also observed in adult estrus cycles

(the equivalent of human menstrual cycles) following perinatal exposure (Markey 2003, Honma 2002).

Specific changes have also been observed in female reproductive tract organs. In the ovaries of perinatally exposed females, alterations in the tissue of the ovaries were seen in early adulthood (Markey 2003). Exposed animals also had an increase in the number of blood-filled ovarian bursae; these are thought to be indicative of advanced reproductive aging. Females exposed to BPA during fetal development had a significant increase in the number of eggs with gross aberrations; when these females were mated, there was a significant increase in the number of chromosomally abnormal eggs and embryos (Susijaro 2007). BPA exposure also alters the growth and development of the vagina and the uterus (Markey 2003, Markey 2005).

v. Cancer

Several recent studies have examined the impact of early exposure to BPA on the development of cancer later in life. One study examined the effects of neonatal BPA exposure on prostate cancer (Ho 2006). Animals exposed to BPA during the neonatal period developed a significant increase in lesions of the prostate when these animals were exposed to hormones in adulthood. These lesions were classified as prostatic intraepithelial neoplasias, a cancer.

My own work in the lab of Drs. Ana Soto and Carlos Sonnenschein has worked to connect BPA exposure to breast cancer in adulthood. Exposure of mice and rats to BPA during early development causes significant changes in mammary gland development. Many of these changes are similar to human risk factors for breast cancer (Markey 2001b, Markey 2003, Munoz de Toro 2005, Vandenberg 2007b, Wadia 2007). In fact, rodents exposed to BPA during the fetal period develop intraductal hyperplasias (precancerous lesions) that progress to carcinomas *in situ* in rats (Murray 2007). Carcinoma *in situ* is a cancer that in women would require surgical intervention. Finally, animals exposed to BPA during development are highly susceptible to low-level carcinogens, developing tumors when unexposed animals do not (Durando 2007, Jenkins 2009).

4. BPA experts produced a consensus statement

In 2006, a meeting of approximately 45 experts in the field of BPA research was organized by the National Institutes of Environmental Health Sciences (NIEHS) with the following purpose: to examine the relevance of ecological, *in vitro* and laboratory animal studies for assessing risks to human health. Together, the scientists at this meeting generated the Chapel Hill Consensus Statement, signed by 38 authors (vom Saal 2007). It stated:

> The published scientific literature on human and animal exposure to low doses of BPA in relation to *in vitro* mechanistic studies reveals that human exposure to BPA is within the range that is predicted to be biologically active in over 95% of people sampled. The wide range of adverse effects of low doses of BPA in laboratory animals exposed both during development and in adulthood is a great cause for concern with regard to the potential for similar adverse effects in humans... There is extensive evidence that outcomes may not become apparent until long after BPA exposure during development has occurred. The issue of a very long latency for effects *in utero* to be observed is referred to as the developmental origins of adult health and disease (DOHaD) hypothesis. These developmental effects are irreversible and can occur due to low dose exposure during brief sensitive periods in development, even though no BPA may be detected when the damage or disease is expressed.

Thus, BPA experts have concluded that there is significant evidence supporting concerns about fetal and neonatal exposure. The data that have been collected in the field of environmental toxicology are sufficient to raise concerns about the potentially deleterious impact of BPA exposure on human development. It would thus be unwise to ignore the incremental evidence stemming from rigorously controlled laboratory experiments alongside the increasing incidence of comparable issues in human populations exposed to these same chemicals during different developmental stages. **All of this evidence should encourage regulatory agencies to apply a preventative approach and thus ban and/or substitute chemicals such as BPA that are likely to be harmful to the normal development of humans and wildlife.**

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